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<u>L24</u>	L23 and (modified or phosphoro\$)	23	<u>L24</u>
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<u>L12</u>	\$5GGGGTGGGG
<u>L11</u>	5\$GGGGTGGGG
<u>L10</u>	\$GGGGTGGGG
<u>L9</u>	GGGGGGGGG
<u>L8</u>	GGGGCGGGG
<u>L7</u>	GGGGAGGGG
<u>L6</u>	5489508 [pn]
<u>L5</u>	L1 and phosphorothioate\$
<u>L4</u>	L2 and phosphorothioate\$
<u>L3</u>	l2 and modified
<u>L2</u>	GGGGTGGGG
<u>L1</u>	GGGGGG

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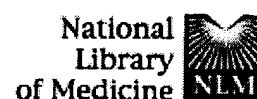
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Telomeres, representing the chromosome nucleoprotein tails, shorten during each cell division due to the inability of conventional DNA polymerases to completely replicate the chromosome termini. When telomeres become critically short, cells are directed to exit from the cell division cycle (replicative senescence). Telomerase is a reverse transcriptase that synthesizes telomeric sequences, thereby prolonging the lifespan of cells. Telomere length and telomerase activity expression vary significantly in different normal somatic tissues and age groups. In many childhood malignancies (ie, acute leukemias and solid tumors), telomere length and telomerase activity of the malignant cell population may be correlated with the disease outcome and thus may be promising tools in evaluating prognosis and monitoring treatment progress. Finally, telomerase inhibition by using several strategies (ie, antisense oligonucleotides) represents a potentially valuable target for antitumor therapy in the near future.

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Telomere Length and Telomerase Activity: Variations With Advancing Age and Potential Role in Childhood Malignancies.

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Abstract:

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